UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLUMBIA

UNITED STATES OF AMERICA	)	
	)	
Plaintiff,	)	Civil Action No. 99-CV-2496 (GK)
	)	
v.	)	Next scheduled appearance:
	)	Trial (ongoing)
PHILIP MORRIS USA INC.	)	
(f/k/a Philip Morris Incorporated), *et al.*	)	
	)	
Defendants.	)	

WRITTEN DIRECT EXAMINATION OF JAMES J. HECKMAN
SUBMITTED BY THE JOINT DEFENDANTS PURSUANT TO ORDER #471

I. INTRODUCTION AND SUMMARY OF BACKGROUND

Q. Please introduce yourself to the court.

A. My name is James J. Heckman.

Q. Where do you live?

A. I live in [DELETED]

Q. Where are you employed?

A. I am employed by the University of Chicago, where I am the Henry Schultz Distinguished Service Professor of Economics in the Department of Economics. I also have part-time appointments at University College London and Peking University, China. I also direct the Economics Research Center at the Department of Economics at the University of Chicago and the Center for Social Program Evaluation at the Harris School of Public Policy at the University of Chicago.

Q. What does the title "Henry Schultz Distinguished Service Professor" refer to?

A. These are actually two titles given to me by the University of Chicago. I joined the faculty of the University of Chicago in 1973 and was named the Henry Schultz Professor in 1985. The Henry Schultz Professorship was awarded in honor of Henry Schultz, a distinguished economist and faculty member at the University of Chicago in the 1920s and 1930s. I received the Distinguished Service Professor title in 1995. A Distinguished Service Professor is a rank above full professor and awarded after a certain amount of time as a professor and in recognition of distinction in one's field.

Q. You also said you are the Director of the Economics Research Center at the University of Chicago. What do you do there?

A. I direct the Center, which organizes seminars and sponsors research across a broad range of topics, such as racial discrimination, industrial organization, macroeconomics, decision-making under uncertainty, among many other topics.

Q. In addition, you also said you direct the Center for Program Evaluation at the Harris School of Public Policy. Would you describe your responsibilities there?

A. I direct research involving social programs, including programs related to GEDs, job training, adolescent risk-taking behavior, early childhood interventions, among many other topics.

Q. Have you provided a copy of your CV?

A. Yes, it is attached as Exhibit JD-012617.

Q. Dr. Heckman, can you tell us a little bit about your academic background?

A. Yes, I received my B.A. (summa cum laude) in mathematics from Colorado College in 1965 and my M.A. and Ph.D. in economics from Princeton University in 1968 and 1971, respectively. Along with my academic tenure at the University of Chicago, I also have served on the faculties of the Department of Economics at Columbia University and at Yale University, where I was the A. Whitney Griswold Professor of Economics.

Q. Would you please describe some of your professional activities?

A. I am a Member of the National Academy of Sciences, a Fellow of the American Academy of Arts and Sciences, a Fellow of the Econometric Society, a Fellow of the Society of Labor Economics, a Fellow of the American Statistical Association and a Senior Research

Fellow of the American Bar Foundation. I also am a Research Associate of the National Bureau of Economic Research.

Q. In addition to your academic appointments, have you advised governments or other world bodies?

A. Yes, in addition to my academic experience, I have served as an advisor to the World Bank, the Inter-American Development Bank, the United States Department of Labor, the Ministry of Fiscal Equity of Argentina, and government agencies in Brazil, Taiwan, South Korea, Germany, Scotland and, next week, Ireland. I have also presented testimony before committees of the United States Congress.

Q. Have you been invited to lecture at different institutions around the world?

A. Yes, I have been asked to give numerous lectures around the world. Some recent major invited lectures include those at Chululongkorn University in Bangkok, Thailand in January 2005, the Toulouse Lecture in Toulouse, France in November 2004, the Hicks Lecture at Oxford University in April 2004, a Keynote Lecture for Ronmiv University at the Great Hall of the People in Beijing, China in December 2003, a Nobel Symposium Lecture in St. Petersburg, Russia in June 2003, a lecture at the Munich Economic Summit in Munich, Germany in May 2003, and the Keynote Lecture at the Tinbergen Centenary in Rotterdam, Netherlands in April 2003, among many others. This week I will be giving the Richard Ely Distinguished Lectures at Johns Hopkins University from April 4 -8. (http://www.econ.jhu.edu/elylectures.html). I am also giving a presentation with other top researchers at a seminar entitled, "The Convergence of Child Development, Neuroscience, and Economics to Guide Early Childhood Policy" at the Biennial Meeting of the Society for Research on Child Development in Atlanta, Georgia on April 9.

Q. Are there any areas in which you specialize within the field of economics?

A. I specialize in the fields of Labor Economics, Applied Microeconomics and Econometrics, which is the application of statistical techniques to economic problems. My life's work has been devoted to developing a scientific basis for social and economic policy evaluation. My work has focused on developing theory and tools for empirically-based decision making for economic and social policy and advancing the methods by which empirical research on these issues is conducted. I also implement these methods and tools in a series of empirical studies.

Q. What do you mean by "empirically-based decision-making"?

A. I'm referring to an approach to policy decision-making that seeks to ensure that policy decisions, when made, are based upon good scientific evidence that the policy, if implemented, will have the intended effects.

Q. Have you published any scholarly articles in these fields?

A. I have published over two hundred articles in scholarly journals and compendia.

Q. And do you serve as a referee or editor for articles published by others in economics?

A. I currently serve as an Associate Editor of the Journal of Labor Economics, Econometric Reviews, and the Journal of Population Economics. I previously have served as Co-Editor of the Journal of Political Economy and as an Associate Editor of Evaluation Review, the Journal of Econometrics, the Review of Economic Studies and the Journal of Economic Perspectives.

Q. Have you received any awards or prizes in the field of economics?

A. In 1983, I received the John Bates Clark Medal awarded biannually by the American Economics Association to the most distinguished economist under the age of 40. In 2000, I was awarded the Nobel Prize in Economics.

II. EXPERTISE OF RELEVANCE IN THIS MATTER

Q. You stated above that your work has focused on "empirically-based decision making for economic and social policy and advancing the methods by which empirical research on those issues is conducted." Could you summarize your research on these matters?

A. Evaluation of social and economic policy is a central problem in the study of economics. Today, the empirical demonstration of causation is a fundamental principle guiding decisions regarding interventions in a variety of areas of social life. Much of my life's work has focused on accurately measuring actual effects of various policies on behavior. In laymen's terms, I have employed econometric and statistical techniques to make careful empirical determinations of whether programs or policies that have been undertaken actually work and how well they work. Thus, my work uses theory and methodology to more accurately draw inferences from observed data.

Q. What is your understanding of the purpose of your testimony here today?

A. I have been asked to discuss the basic elements and processes of empirical scientific investigation that have been widely adopted for decision-making regarding various interventions. Specifically, I have been asked to evaluate the opinion and testimony of Dr. Eriksen concerning the relationship between advertising and youth smoking in light of these principles. I have also been asked to evaluate the evidence that Dr. Eriksen relied upon for rendering his opinion that

defendants' advertising and marketing are "substantial contributing factors" to youth smoking initiation.

Q. How is your academic work and research related to the question of whether advertising cause youth smoking?

A. I have written extensively on how to model choices and make causal inferences concerning what's referred to in economics as "individual economic behavior," or more plainly, human behavior. Much of my research has dealt with the evaluation of social policies that are intended to affect human behavior. The same reasoning and methodology I would use to evaluate a job training, welfare program, or the returns to early childhood education, I apply to the studies presented by Dr. Eriksen as evidence that advertising causes young people to smoke. The evidence in this case related to advertising and youth smoking is based on interpretation of observed data – thus the issue is the same, whether it is a social policy or an "action" such as advertising that is experienced by people. The role of choice and individual characteristics needs to be properly understood and accounted for in models attempting to estimate the effect of a policy or action. Therefore, my work on policy evaluation shows the importance of sound theory and methodology in practice and will highlight the issues of relevance for the studies presented as evidence by Dr. Eriksen.

Q. Is the work for which you received the Nobel Prize relevant to your interpretation of the evidence concerning the relationship between advertising and youth smoking?

A. Yes. The Nobel Prize website describes the award for "development of theory and methods for analyzing selective samples." (http://nobelprize.org/economics/laureates/2000/). The presentation speech also described my work in this way: "The methods you developed, together with new data sets and powerful computers, made it possible to study individual

economic behavior in a statistically correct way. *In your own applied research you demonstrated how solid empirical knowledge can help address important social problems*." (http://nobelprize.org/economics/laureates/2000/presentation-speech.html, emphasis added)

Q. Are you considered a leading world expert at how to evaluate causes of individual economic behavior?

A. Well, I certainly have a lot of experience working on the topic. I have written extensively on the subject and have been widely cited as having developed and employed sound scientific and empirical techniques to evaluate individual economic behavior and programs designed to affect individual economic behavior. My research has focused on policies that have significant social consequences: policies involving discrimination, welfare, youth behavior, education, and job placement for the unemployed. Furthermore, as I noted previously, the Nobel Prize committee cited my work on program evaluation as an important contribution to the field of economics, as well as other social sciences. Accurate evaluation of public programs requires thorough understanding and careful measurement of program effects on individual behavior. The theory and methodology I have developed have been applied in many academic fields. I have been involved in evaluating various intervention programs in many countries as well as teaching theory and proper methodology to students and analysts around the world. Recently, I gave a talk in Thailand touching on many of the conceptual and methodological issues that are at the heart of this case – reliable causal inference and sound statistical analysis. I have helped launch the rigorous empirical study of evaluation of social policy in that country and plan to do so in China under the term of my Chang Jiang / Yangtze River Professorship.

Q. Could you explain more specifically how policymakers benefit from a careful empirical analysis of a particular program's effects?

A. With limited funds available for social programs, identifying and implementing those programs most likely to help the intended beneficiaries obviously is important. The simple fact is that many programs or policies that people expect to be helpful turn out to have little or no social benefit when proper analysis is done; indeed, some well-intended programs implemented by well-meaning agencies or institutions have actually turned out to have *harmful* effects. If the true effects of a policy on its targeted beneficiaries are not well understood, programs may be poorly designed.

Q. Are you saying that decision-makers must have an empirical demonstration of causality before making decisions relating to programs or interventions?

A. No. Decision-makers often do not have definitive demonstration of causation before implementing a particular program or intervention. However, careful consideration should precede claims about likely effects of policies or interventions without empirical and theoretical support. Indeed, before claiming that a policy or program has actually had an effect, careful empirical investigation would be required. Dr. Biglan agrees that empirical evaluation of the effects of interventions and programs is important as well. "Such long-term follow-up evaluations are costly, but important, to do so that policymakers can make informed choices about how to use often scarce resources." (Biglan, et al., *Helping Adolescents at Risk: Prevention of Multiple Problem Behaviors*, 2004, p. 126). He further explains that "Without comparative data, however, we will never know their real benefits or costs. In addition, the cost of an ineffective intervention that continues due to political or other pressures in the absence of evaluation is high, because it robs children of the chance to have experiences that might really make a difference in their lives." (Biglan et al., 2004 p. 126)

Q. Previously you mentioned that some well-intended programs have not had the desired effect. From your own research can you give an example of a policy that people thought would be beneficial, but that you showed through careful analysis was not?

A. The government implements, and has implemented in the past, a variety of job training programs intended to improve the employment prospects of the currently non-employed. Government officials wish to evaluate these programs, to see whether they increase employment rates. They compare the job market performance of program participants with that of a random sample of non-participants with similar "observable characteristics": for example, education, test scores, race, age, and so forth. They find that program participants are significantly more likely to have found a job. They therefore conclude that programs with these characteristics are successful.

Q. Are these programs successful?

A. I analyzed these programs and discuss my views in Heckman, et al., *Handbook of Labor Economics*, Vol. 3:1865-2097 (1999). The main thrust of my research has been to show that such a conclusion is often premature when one takes a superficial look at the data. The government officials face a very complex task, solving what has been termed the "evaluation problem." At its base is the simple fact one cannot observe counterfactuals; i.e., how the individuals who entered the training program would have done on the job market had they not been trained, or how the comparison group would have done had they been trained. Suppose that people who join such training programs tend to be more motivated, ambitious, or capable than non-joiners, and that these differences cannot be captured by available statistical measures such as age or education. Then simply comparing employment outcomes across these two groups, controlling only for age and education, will vastly overstate the effect of the training program—

the program participants would have done better than the non-participants *even without the training*. In other words, there is a selection problem—those who join training programs differ systematically from those who do not in unobservable, but relevant, ways. I developed econometric techniques to deal with these selection problems. These techniques are now a standard aspect of any empirical research involving the study of human behavior, and are cited in textbooks on the topic, such as Greene, *Econometric Analysis*, 2005; and Judge, et al., *The Theory and Practice of Econometrics*, 1985.

Q. What did you find once you employed this methodology to the study of job training programs?

A. In the context of job training schemes, I found that once the proper econometric techniques and more rigorous methodology had been employed, the public job programs I studied turned out to have a very small effect (if any) on labor market performance, as discussed in the aforementioned *Handbook of Labor Economics*.

Q. Have others replicated your findings?

A. Yes, our findings were later replicated by John Martin and David Grubb in an article entitled "What works and for whom: A review of OECD countries' experiences with active labour market policies" in the Swedish Economic Policy Review, 8: 9-56, 2001. They found that some programs appear to yield *negative* rates of return when the effects are compared to program costs. In a similar vein, subsequent studies have concluded that "displacement effects"—newly trained workers simply displace non-trained workers—may be sizeable, so that total employment is only marginally affected by training programs, again, contrary to expectations, as discussed in Davidson and Woodbury, "The Displacement Effect Of Reemployment Bonus Programs," Journal of Labor Economics, 11: 575-605, 1993, and

Heckman et al., "General Equilibrium Treatment Effects: A Study of Tuition Policy," American Economic Review, 88: 381-386, 1998.

Q. What has been your advice to government bodies or other institutions contemplating public policy changes intended to impact human behavior?

A. It is often very difficult to predict *ex ante* what the effect of a given policy will be. Predicting how people will react to particular policies requires very careful thought. Indeed, some of the most well-meaning policies have produced results directly opposite to what was sought. The fact is that policies have often changed the incentives of the affected parties in unanticipated ways, with unexpected results.

Q. Can you provide some examples?

A. One good example involves child-proof safety caps. Congress passed the Poison Prevention Packaging Act in 1970 in an attempt to reduce the number of poisonings of small children. That is obviously a worthy goal. Under the new law, manufacturers were required to design and employ packaging that was difficult to open. The objective was to reduce the likelihood that young children would ingest harmful substances. Yet, contrary to expectations, Professor Kip Viscusi found evidence that the Act actually may have *increased* the number of poisonings of young children. Professor Viscusi suggested at least two possible explanations: First, because the bottles were more difficult to open, they were left open more frequently (by older people with arthritis, for example); and second, parents may have been lulled into thinking the caps made the containers completely impossible for a child to open (which wasn't true), and therefore took less care to place containers out of reach. These effects were certainly not intended by the designers of the law, but resulted from people changing their behavior in response to the law in "reasonable" ways that reduced or defeated the intended effect of the law.

These findings are discussed in Viscusi, "The Lulling Effect: The Impact of Child-Resistant Packaging on Aspirin and Analgesic Ingestions," AER Papers and Proceedings, 74: 324-327 (1984), and Viscusi, "Consumer Behavior and the Safety Effects of Product Safety Regulation", Journal of Law and Economics, 28: 527-554 (1985).

Q. Are there any other examples?

A. Yes. There are many more examples. Rent control laws intended to help the poor and struggling have led to significant shortages of rental apartments, from which the poorest and least-educated suffer disproportionately (better educated renters quickly learn how to play the system) (Mankiw, *Principles of Economics,* pp.115-117, 1998.) Federal deposit insurance, intended to protect small savers, gave bankers the incentive to play roulette with their depositor's money, and the S&L crisis of the 1980s was the result. (Jaffee, "Symposium on Federal Deposit Insurance for S&L Institutions," *Journal of Economic Perspectives,* Vol. 3, no. 4, 1989) The basic message is that one must carefully consider behavioral responses to policy changes before implementing any particular policy; otherwise these policies can often lead to unintended consequences.

Q. What can your work tell us about studies measuring the effects of advertising on teenage smoking?

A. Just as the application of sound scientific and empirical techniques is crucial for accurately evaluating program outcomes, the same is true for evaluating the studies relied upon by Plaintiff's experts relating cigarette advertising and marketing to youth smoking. These techniques can be used to see if the factor of interest has been isolated sufficiently to estimate its effect on otherwise similar groups.

Q. Without discussing any of the evidence relied upon by Dr. Eriksen, can you give a hypothetical example of how the selection problems you identified above might be relevant to the study of advertising and youth smoking?

A. Suppose, for example, that it is empirically established that 15 year olds who wear t-shirts with cigarette logos are more likely to smoke later in life than teenagers who do not wear t-shirts with cigarette logos. A naïve conclusion would be that wearing t-shirts with cigarette logos (or attention to cigarette advertising generally) leads to smoking. My research on the selection effect indicates that one must think about the question more carefully. Teenagers who wear t-shirts with cigarette logos may very well differ in important but unobservable ways from those who do not, and it may be that these unobservable differences that make the t-shirt wearers more likely to smoke later in life. For example, the wearing of t-shirts may reflect an unobservable tendency to rebel or an affinity with smokers that is completely unrelated to cigarette advertising. Indeed, the t-shirt wearers may have been more likely to smoke regardless of whether they had been exposed to any tobacco promotional items or advertising. In other words, there is a selection problem. We cannot simply assume that teenagers who wear t-shirts with logos are in all ways identical to those who do not, controlling for observable characteristics. To do so may lead to the same erroneous conclusions as in the job training program example I discussed.

Q. How would you overcome this problem?

A. To disentangle the various possible causal effects, and thus overcome the selection problem, the statistical techniques that I helped to develop and discussed previously in this testimony, must be employed—simple correlations or regressions will produce misleading results.

Q. Does any of your academic work and research specifically address issues related to the behavior of children and adolescents?

A. Yes, much of my work over the past twenty years has been focused on investigating skill and ability formation, a topic that is highly relevant to labor economics. More recently, I have been examining the causes of various types of youth behavior, such as schooling, teen pregnancy, crime, drug use and smoking. My current research, drawing on the literature in economics and other pertinent fields, investigates common factors that play powerful roles in explaining a variety of behavioral problems that emerge in the adolescent years.

Q. You mentioned your research involved studying the causes of youth behavior. Could you describe the behavior you're researching?

A. Yes. While I have been studying, broadly speaking, how human skills and ability are formed, I have been studying how to create healthy children and how to prevent them from entering the underclass. Growth of the underclass is a serious problem in the United States and in countries around the world. This study involves examining the sources of human differences and the causes of pathological, abnormal behavior. I look at early differences in children in terms of cognitive and noncognitive skills and ways to prevent or limit problems that arise later in life. These problems may include crime, violence, children born out of wedlock, as well as lower economic success later in life. Some of the associated behaviors include involvement with drinking, smoking, drugs and other risky behaviors.

Q. What has your research shown?

A. My research suggests that common factors related to ability ("cognitive factors") and to self-regulation and self-perception ("non-cognitive factors") play powerful roles in explaining a

variety of behavioral problems that emerge in the adolescent years. My work goes further than the current literature by pointing out that these abilities or factors are determined at very early ages of the child, long before adolescence. One of the major determinants is family -- family environments, as well as genetic factors. In "family environments" I include the in utero environment created by the mother's own behavior, such as her own smoking, drinking or stress factors operating on the fetus. The important lesson that is emerging from a variety of studies is that abilities of many sorts are shaped at early ages and they greatly affect child development and child choices to participate in risky behaviors. Some examples of such studies are: Bowles et al., "The Determinants of Earnings: A Behavioral Approach," Journal of Economic Literature, 39(4): 1137-1176, 2001; Heckman and Rubenstein, American Economic Review, 91(2):145:149, 2001; Knudsen, "Sensitive Periods in the Development of the Brain and Behavior," Journal of Cognitive Neuroscience, 16(8): 1412-1425, 2004; Turkheimer et al., "Socioeconomic Status Modifies Heritability of IQ In Young Children," Psychological Science, 14(6): 623-628, 2003.

Q. Have you published papers on this research?

A. Several papers have been accepted and are due to be published soon. One recent article I co-authored entitled "Interpreting the Evidence on Life Cycle Skill Formation" is due to be published in the *Handbook of the Economics of Education*. Cunha, Heckman, et al., "Interpreting the Evidence on Life Cycle Formation," (revised March 28, 2005) (JD-013265). The goal of this article is to "provide a theoretical framework for interpreting the evidence from a vast empirical literature, for guiding the next generation of empirical studies, and for formulating policy. Central to our analysis is the idea that childhood has more than one stage, and that policies need to be tailored to each one. We define the concepts of self productivity and complementarity of human capital investments and use them to explain the evidence on skill

formation. Together they explain why skill begets skill. Skill formation is a life-cycle process. It starts in the womb and goes on throughout most of the adult life. Families and firms have a roll in this process that is at least as important as the role of schools. There are multiple skills and multiple abilities that are important for adults' success. Abilities are both inherited and created, and the traditional debate about nature versus nurture is scientifically obsolete. The returns to investing early in the life cycle are high." We find that the same set of factors can explain many "risky" behaviors, and that early remediation efforts can be effective in improving outcomes. Other recent articles addressing similar issues are "Lessons from the Technology of Skill Formation," due to be published by the New York Academy of Sciences, and "The Productivity Argument for Investing in Young Children," Working Paper 5, Invest in Kids Working Group of the Committee for Economic Development, co-authored with Dimitriy Masterov.

Q. Have you presented these findings concerning the importance of early skill development and investment in programs to assist very young children at conferences or other public speaking engagements?

A. Yes. I've presented my findings at many venues, including the NIH, the Committee on Economic Development, the University of Wisconsin, University College London, the Public Economic Theory Meetings in Beijing, China in August 2004, the Toulouse Lecture Series in November 2004, the University of Montreal in 2004, and the Chicago Federal Reserve Seminar on Labor Economics April 1, 2005, among others. I also will be discussing my findings at the Johns Hopkins Ely Distinguished Lectures Apr. 4 – 8, and at the Geary Lecture in Dublin, Ireland at Trinity College April 22, 2005.

Q. Could you describe some of your more recent work related to youth skill and ability formation?

A. In a working paper, Jeffrey Smith and I analyze the U.S. Job Training Partnership Act (JTPA), which is intended to help disadvantaged youths earn higher wages. (Heckman and Smith, "The Sensitivity Of Experimental Impact Estimates: Evidence From The National JTPA Study," NBER Working Paper 6105, 1997). An experimental evaluation had concluded that the JTPA was of little help to either males or females; indeed, the evaluation stated that the earnings of young males were *reduced* by JTPA. These conclusions were being used to justify cuts in the JTPA budget. Jeffrey Smith and I find, by contrast, that the experimental evaluation's results are very sensitive to precisely how the experiment was structured. Indeed, taking other factors into account, we conclude that the overall assessment of the program is much more positive. I also have working papers in which my co-authors and I analyze methods for estimating the returns to schooling—a policy question of great relevance to young people. These papers include Heckman, Tobias, and Vytlacil, "Simple Estimators For Treatment Parameters In A Latent Variable Framework With An Application To Estimating The Returns To Schooling," NBER Working Paper 7950 (2000); Heckman and Rubinstein, "The Importance Of Noncognitive Skills: Lessons from the GED Testing Program," AEA Papers and Proceedings, May 2001; Cawley, Heckman and Vytlacil, "Three observations on wages and measured cognitive ability," Labour Economics 2001; Carneiro and Heckman, "Human Capital Policy," in Discussion Paper No. 821, July 2003; Hansen, Heckman and Mullen, "The effect of schooling and ability on achievement test scores," Journal of Econometrics, 121:39-98, 2004; and Cameron and Heckman, "The Dynamics Of Educational Attainment For Black, Hispanic, and White Males," Journal of Political Economy, June 2001, 109(3), 455-499. As I mentioned earlier, I have papers in which I

investigate policies intended to promote education and skill development at various stages of life, and conclude that the most effective policies are those that focus on helping people while they are still young. These findings are summarized in a publication by the Ounce of Prevention Fund and the Harris School of Public Policy, entitled "Invest in the Very Young." (www.ounceofprevention.org/downloads/publications/heckman.pdf.)

Q. Are you relying on these newer studies in forming your opinions in this case?

A. My opinions concerning the relationship between advertising and smoking were formed based upon the evidence available to me at the time I wrote my report in this case. Nothing I have learned, either in my own academic research or research of others or in the studies and evidence offered by Dr. Eriksen has changed my original conclusion that the evidence does not support a causal association between smoking and advertising. If anything, my own more recent academic work, and that of others, has reinforced my original findings.

III. SCIENTIFIC MODEL OF CAUSALITY

Q. The issue you've been asked to address involves the relationship between advertising and smoking by youth. Can researchers use scientific methods to find causal relationships when modeling human behavior?

A. Yes, scientific methodology can be applied here as in other areas. Researchers develop a hypothesis that they'd like to test. They design a model or experiment that will isolate the factor they'd like to test, they gather appropriate data and they estimate the effect. They test how sensitive their results are to the assumptions and specification of the chosen model. JDEM 010352, 010353, 010381, 010354, 010355, 010356.

Q. Is this the same methodology that is applied in chemistry experiments, or in epidemiological studies to find causal relationships?

A. There are indeed many similarities. In each case, the goal is the same – to isolate the factor being tested and measure the effects of changes in this factor on outcomes. As in all types of modeling, from the effect of sunlight on the growth of a bean plant, to the effect of a drug on a disease, to the effect of a social program like welfare reform on the employment of welfare mothers, the researcher attempts to isolate the factor being tested. In the example of testing the effects of sunlight on the growth of a bean plant, there probably are a limited number of factors to "control" for in order to isolate the effect of the sunlight. These may include the amount of water, fertilizer and temperature. When evaluating the effects of a drug on a disease, the number of factors to deal with rises. These may include the presence of other diseases, whether people follow the drug regimen or are administered the drug, overall health, diet, stress, age, race, and the like. When trying to measure the effect of a social policy, such as welfare reform, the number of factors, and interactions of factors, both observable and unobservable rises. JDEM 010357, 010358, 010359.

Q. What are the important differences in studying human behavior, as opposed to physiological events?

A. Causal models in epidemiology and statistics focus on outcomes of treatment. Econometric models not only model the causes of outcomes, but also the role of choice in the treatment actually chosen. Econometric models also consider the relationship between the factors determining outcomes and the factors producing choices. Because choice plays such an important part in the treatment experienced by individuals, neglecting to model choice may result in faulty inferences and misattribution of the cause of an effect. The complexity of modeling

human choice requires more elaborate theory and modeling than is envisioned in the current literature on causal inference in epidemiology and statistics. The econometric literature has advanced to include sophisticated methods as well as theory to make causal inferences regarding human behavior. JDEM 010367.

Q. Why is it important to understand why people do what they do?

A. The goal of econometric analysis, like the goal of all scientific study, is to model phenomena at a deep level and to understand the causes or mechanisms producing the effects and the choices people make. Once this is done, one can use the empirical versions of the models to forecast the effects of interventions never previously experienced, to calculate a variety of policy options and to use scientific theory to guide the interpretation of the evidence.

Q. Given all these issues, can you describe criteria you believe are necessary to find causal influences in studies of human behavior?

A. In economics, there are well-reasoned principles that are taught, and that I teach to students. First, researchers should have a well-specified model of outcomes and choices of outcomes. A well-specified model should be based in sound reasoning and theory and explicitly state assumptions that are being made. Assumptions used to construct counterfactuals potentially affect the interpretation and the generalizability of the results. A well-specified model should distinguish among alternative explanations for the same phenomenon. A model of human behavior must not only model the outcomes, but the choices that give rise to the outcomes, and the set of factors that drive these choices, that may have led to these outcomes. This is important because researchers observe the outcomes of choices made. Therefore, it is important to distinguish the effects of the factors that led to the choice from the effects of the choice itself. This is no different from any other scientific endeavor in which the researcher tries

to isolate the effect of one factor by comparing groups that are similar along all other relevant dimensions. It is just that with the study of human behavior the problem with multiple other factors influencing the factor that you're trying to isolate is more complicated and has to account for choices. JDEM 010367.

Q. Are there any other principles to which researchers should adhere when analyzing causal relationships in studies of human behavior?

A. As in a study in any field, rich data is desirable. Ideally, researchers would like data that accurately measures the factor being tested, or a good proxy for it, and data on relevant other factors that help isolate the effect of the factor being studied. The use of appropriate empirical methodology, including robustness (or sensitivity) tests, provides a sound basis for interpreting results. Ideally, replication of results by other researchers would provide additional support for a sound analysis from which one may draw causal inferences.

IV. SUMMARY OF TASK AND CONCLUSIONS

Q. Dr. Heckman, for this case, you stated that you were asked to assess the evidence presented by Dr. Eriksen related to the effect of tobacco company advertising on youth smoking. What work have you undertaken in order to do that?

A. I have reviewed reports and testimony of various experts, with particular focus on the testimony and evidence presented by Michael Eriksen related to youth smoking. I also have reviewed reports on smoking by the Surgeon General of the United States, the economics and public health literatures that address smoking behavior and cigarette marketing, a large body of the literature on adolescent risk-taking behavior, the literature that evaluates the effect of intervention programs on smoking behavior, and survey data related to both youth and adult smoking and related behaviors. When I use the term marketing, I refer to "non-price" related

marketing, such as advertising, t-shirts and sport sponsorships, which have an image component. Price-related marketing, such as coupons, price discounts or package give-aways have qualitatively different effects, and well-established evidence in the literature supports a causal relationship between price and cigarette consumption, as is true with most goods and services.

Q. Before I ask you to explain your conclusions to the court in detail, let me first ask: are you aware of the following statements the government made in its interim summation on February 24, 2005:

> "Dr. Eriksen testified under oath, specifically, that there is no scientific debate today about the causal relationship between marketing and youth smoking behavior." (2/24/05, p. 14253 at 16-18)
>
> "If you frame the question properly, is there a causal relationship between marketing and youth smoking initiation, the answer will be yes, and we heard that from several experts. The United States has proven that causal connection exists." (2/24/05, p. 14255, at 1-4)

A. Yes.

Q. Do you agree that there is no scientific debate today about the causal relationship between cigarette marketing and youth smoking behavior?

A. No. I do not agree with the assessment that there is no debate in the scientific community as a whole about the causal relationship between cigarette marketing and youth smoking behavior. Actually, this debate is explicitly recognized in the major reviews of the economics literature on advertising and smoking, one by Martyn Duffy , "Econometric Studies Of Advertising, Advertising Restrictions and Cigarette Demand: A Survey," *International Journal of Advertising,* 1996, 15: 1-23 (JD-062203) and the other coauthored by the government's expert

Frank Chaloupka (Chaloupka and Warner, "The Economics Of Smoking," NBER Working Paper #7047, March 1999).

Q. Are you also aware of the government's claim in its interim summation that:

"The scientific community agrees that cigarette marketing is a substantial contributing factor to youth smoking initiation and continuation." (2/24/05, p. 14256, at 2-5)

A. Yes.

Q. Have you also read Dr. Eriksen's testimony during which he opines that cigarette advertising and marketing are substantial contributing factors to youth smoking initiation and continuation, including the following:

". . . marketing, while not the only factor, is a substantial contributing factor . . ." (2:1-2)

". . . advertising and promotion affects smoking behavior." (55:22-23)

". . . there is a positive relationship of advertising and promotion on cigarette consumption." (55:24)

". . . it is clear that the preponderance of the evidence suggests that cigarette marketing influences adolescent smoking behavior." (56:1-2)

". . . clear and compelling evidence . . . that advertising and promotion influences each of the factors that lead directly to adolescent tobacco use . . . " (58:2-3)

A. Yes.

Q. Has it been established that cigarette marketing is a substantial contributing factor to youth smoking initiation and continuation?

A. I take the government's experts to be saying that by marketing being a "substantial contributing factor," they mean that by decreasing cigarette marketing the propensity of youth smoking initiation would in turn decrease in a nontrivial way. In other words, a causal factor has a substantial impact. It is this point with which I principally disagree. The evidence that the government's experts rely on does not demonstrate that tobacco company advertising is a causal factor for youth smoking initiation or for continued use of cigarettes, nor does other evidence that I am aware of, not relied upon by the government's experts but relevant to the question at issue, support that assertion. However, if the government experts are saying only that a statistical association exists between youth smoking propensity and cigarette marketing and that this statistical association provides no reliable basis from which to draw conclusions on the effects of reducing tobacco company advertising on youth smoking initiation, then I agree with that statement.

Q. Is there a distinction made in the science of economics between a statistical association and causation?

A. Yes, it is a central distinction, not only in economics, but in many scientific disciplines, such as sociology and epidemiology, that rely upon statistical models to obtain estimates of risk. In economics, the test of whether an input is a "causal" factor considers whether an outcome would be changed if that input, *and that input alone*, were varied. In this case, what I understand the government to be claiming is that if there were less tobacco company advertising, everything else the same, fewer adolescents would start smoking, and/or maybe they would be more likely to quit. That is exactly the definition of a "causal factor" in economics -- it is a factor that, all else constant, can cause a change in outcomes. An association is a statistical relationship between or among variables that does not require any causal implications.

Q. Have you reviewed Dr. Eriksen's statements using various terms to describe the relationship between cigarette marketing and youth smoking?

A. Yes, I have.

Q. In your view, what is it that Dr. Eriksen has established empirically: causation or association or both?

A. Whatever term that is used to describe the relationship between cigarette marketing and youth smoking, the evidence that has been provided does not establish a causal relationship. The associations in the studies presented far from establish a causal relationship.

Q. What would a study need to establish a reliable causal inference?

A. It would include the following elements: (1) valid measurements of both outcomes and alleged causes as well as other probable causes, (2) a clear argument demonstrating that other potential causes, other than the one in question, have been accounted for, and (3) replicability (other researchers can follow and duplicate the analysis).

Q. Dr. Heckman, you have just defined a set of characteristics for a study or a group of studies to provide a reasonable basis for reliable inferences. Is it possible for a study or a set of studies to provide reliable evidentiary support for a causal inference if these characteristics are not perfectly met?

A. Yes. In the real world, researchers often encounter less-than-ideal data in conducting their analyses. A major portion of my research over my life has been devoted to developing tools and techniques for dealing with less-than-ideal data. Proper theory and statistical methodology can be employed to compensate for the inherent shortcomings of the data. With less-than-ideal data, careful empirical methodology often can be used to produce empirical results that provide

reliable evidence, provided it is suitably qualified. A cornerstone of careful empirical methodology is sensitivity analysis that examines how robust the conclusions of a particular study, or a particular analysis, are to alternative specifications including choices of variables, functional forms, and estimators.

Q. In order to make well-founded causal inferences in the context of tobacco company marketing and youth smoking initiation, is it necessary to conduct a randomized controlled experiment?

A. No, it is not. There are many instances in economics and in other fields where scientists have developed well-founded causal conclusions based on study designs other than randomized controlled experiments. Instead, scientists have relied on randomization that occurred without the imposition of a controlled experimental design, such as with observational studies. For example, in the case of determining the effects of cigarette marketing on youth smoking initiation, a researcher could identify and compare two samples of adolescents, one with high exposure to advertising, and another with lower exposure to advertising, *holding constant for each group other factors which may affect the decision to smoke (i.e. preferences for smoking).*

Q. Then your basis for concluding that the government's experts' evidentiary support for the conclusion that cigarette marketing causes youth smoking initiation is unreliable is not based on the absence of a randomized controlled study?

A. Correct. That is not the basis for my disagreement.

Q. What is your basis for saying that Dr. Eriksen has not provided reliable evidence to support the conclusion of a causal link between cigarette marketing and youth smoking initiation?

A. Dr. Eriksen cites studies that are flawed in terms of both the methodology employed and the data used as the basis for his expert opinions about causality. These flaws have nothing to do with the absence of a randomized controlled experiment. Of course, if valid, experimental data were available, it would be useful in establishing or refuting causal claims.

Q. Are these flaws minor such that one could still glean useful information for determining if there is a causal relationship between cigarette marketing and youth smoking initiation?

A. These flaws are fundamental and important enough to render these studies potentially very misleading for addressing this question of a causal relationship between cigarette marketing and youth smoking initiation.

Q. Now given these clarifications, can you summarize your basic conclusions concerning the effect of tobacco company marketing and youth smoking?

A. I have two basic conclusions. First, as I have already stated, the evidence on which Plaintiff's experts rely does not provide a reliable basis for concluding that tobacco company marketing has caused youth smoking initiation. Second, the available evidence in the developing literature on adolescent risky behavior, including smoking, supports a multi-causal model for youth smoking, as many potential causal factors have been investigated in the literature. These factors include price, parental influences, risk preferences, peer influences, access, and advertising, among other things. In my judgment, there exists an empirical basis which supports a causal relationship between some of these other factors and youth smoking, such as between price and youth smoking. However, this type of solid basis of evidence is lacking for the relationship between cigarette marketing and youth smoking.

V. SCIENTIFIC EVIDENCE DOESN'T SUPPORT A CAUSAL LINK BETWEEN TOBACCO COMPANY MARKETING AND YOUTH SMOKING

Q. What is your opinion as to whether the longitudinal studies in the "Cochrane Review" (Lovato, et al., Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours (Review)), The Cochrane Database Of Systematic Reviews (JD 013159), cited by Dr. Eriksen in his direct testimony (p. 79-80) support a conclusion that cigarette marketing is causally related to youth smoking initiation?

A. No, they do not. The studies cited in the Cochrane Review are flawed in terms of both the methodology employed and the data used for the purpose of determining a causal link between cigarette advertising and youth smoking initiation. Therefore, these studies do not provide a reliable empirical foundation from which to draw conclusions on this causal link.

Q. Before I ask you about the individual studies, what is the "Cochrane Review"?

A. The first I heard of the Cochrane Review was in reading Dr. Eriksen's direct testimony. I saw from their website that they are some type of research organization that attempts to analyze bodies of evidence concerning various medical issues.

Q. Is it a reliable source of information on the causal relationship between advertising and youth smoking?

A. I have never relied on their studies and have not encountered it in my scholarly work.

Q. Before you individually discuss the articles cited in the Cochrane Review and relied upon by Dr. Eriksen, would you briefly review the central reasons why you believe those studies do not provide an evidentiary basis from which to draw reliable conclusions about a causal link between cigarette advertising and youth smoking initiation?

A. Yes. There are three basic reasons why I conclude that these studies are flawed and therefore do not provide any reliable evidence on the effects of advertising on youth smoking initiation. The first problem is that these studies do not develop empirical causal models that are theoretically and statistically robust. By that, I mean they do not entertain or test against plausible alternative explanations and specifications of their own data and models.

Q. Could you explain this first problem in laymen's terms?

A. Yes. First of all, the causal links between possible or plausible factors and smoking outcomes are not carefully delineated and adequately investigated. If one does not carefully define the structure of the relationships being investigated in a particular setting—specifically regarding the direction of causality and the factors potentially involved—one can make egregious errors in deriving causal inferences from correlation evidence. A well publicized example involves a study published in the May 13, 1999 issue of *Nature*, a highly regarded scientific journal. (Quinn et al., "Myopia and ambient lighting at night," Nature, 399: 113-114, May 13, 1999). (JD-013261). The study found that babies younger than two years of age who slept with a nightlight on were more likely to become myopic later in life. Because the investigators had not developed a well-founded (scientifically or biologically based) model, they had no explanation for this "surprising" finding. What followed was much ad hoc "theorizing" about the effects of nightlights on infants' eyes. However, a year later, *Nature* published a second study contradicting the conclusions of the first. (Zadnik et al., Myopia and ambient night-time lighting, Nature, 404: 143-144, March 9, 2000). (JD-013262). This second study acknowledged that there was a correlation between nightlight use and childhood myopia, but developed a more complete model to explain it—nearsighted parents were more likely to employ nightlights than were parents with good vision. In other words, the reason babies exposed to

nightlights were likely to become nearsighted had nothing to do with nightlights and everything to do with nearsighted parents. The first study had erred in failing to identify the direction of causality and the factors underlying the decision to turn on a nightlight and failing to present readers with a coherent explanation of its "findings." A carefully implemented causal model, rooted in biology and physiology, would have accounted for the fact that nearsighted parents are more likely to use nightlights (because they have more trouble seeing) and that the children of nearsighted parents are more likely to become nearsighted (because such traits are inherited). Thus, it would avoid misattributing a causal relationship between nightlights and nearsightedness, even though a strong correlation may exist in the data. This is precisely a problem with the studies cited by Dr. Eriksen, as these studies do not establish the difference between correlations and causes.

Q. What is the second problem with the studies?

A. Second, these studies ignore well-established principles of statistical analyses of human behavior. Specifically, these studies ignore the consequences of human choice for the validity of their statistical analyses. Their procedures do not capture, and indeed, do not entertain the possibility of deliberate human responses to complex real world stimuli, such as advertising. Thus, the studies ignore commonly accepted techniques and methods for conducting statistical analyses of human behavior, such as the method of control functions, the method of instrumental variables and other modern methods of statistical inference in causal models.

Q. Would you please tell the Court your second reason for why you believe the studies put forth by Plaintiff's experts are flawed in layman's terms as well?

A. Yes. The authors of the studies cited by the Plaintiff's experts have ignored simple and commonly accepted principles of causal inference by not accounting for self-selection in their

studies. Specifically, the "receptivity" measure in these studies, represented by the government's experts as a proxy for participants' advertising and marketing *exposure*, is plausibly related to already existing unobserved preferences to smoke among the adolescents studied. Therefore, even without a formal structural model, careful reasoning suggests the use of a variety of statistical techniques to mitigate the effects of self-selection on these empirical analyses.

And here is a simple explanation of this problem. Since inferences in these studies are based on differences between groups, namely between the high receptivity group and the other group(s), it is important to make sure, as well as can be expected with the available data, that these groups are similar *in every way except* with respect to the treatment variable of interest. The studies have not credibly controlled for other plausible factors. For example, one of the receptivity measures used in the studies is the possession of tobacco promotional items. It seems likely that participants who have greater preferences for smoking would seek out and obtain more tobacco-related items, even if the items themselves have no independent causal effect on their desire to smoke. As a result, a simple correlation between the possession of such smoking related items and future smoking behavior does not indicate that the items *caused* smoking. In other words, participants who already are more likely to smoke would be more likely to be classified as high-receptivity individuals, all else equal, given the study designs employed. Therefore, observing a correlation between the receptivity measure and smoking uptake is not reliable evidence of any causal effect.

Q. What is the third problem with these studies?

A. In addition to the preceding methodological flaws, these studies employ questionable measures of the theoretical constructs that are purportedly investigated. Specifically, all the "longitudinal" studies listed in the Cochrane Review use measures for advertising and marketing

exposure that have not been shown to be related to the actual advertising and non-price marketing exposure experienced by the participants of these studies.

Q. Again, can you briefly describe the third problem in laymen's terms?

A. Yes. The fundamental problem is that the key variable of interest may not have any relation to participants' actual exposure to advertising. That is, the receptivity variables have not been demonstrated to vary across sample participants according to variations in advertising and non-price marketing campaigns. Among econometricians, this is commonly called a bait-and-switch strategy. This is a common rhetorical device in statistics, namely to call a variable something you want to measure and then actually measure something very different. But you get the reader hooked into your interpretation of the data by using one word to describe a very different concept from what is actually used in the statistical analysis. JDEM 010373, 010372. Put another way, receptivity is being used as a proxy for advertising exposure, however that proxy has not been validated.

In this case, ownership of a tobacco item or receptivity measures are related to later smoking propensity. Ownership of an item related to smoking is equated to exposure to general tobacco advertising campaigns. This association confounds personal preferences with exposure to tobacco company advertising campaigns. A built-in correlation between the receptivity measures and smoking may exist because it may simply capture preferences, as adolescents with greater preferences for smoking are more likely to smoke. Receptivity and the variable it is supposed to measure--advertising exposure--are fundamentally distinct concepts. Plaintiffs have not produced direct evidence that advertising campaigns raise smoking among youth. That would require that they compare different campaigns directed to similar persons and measure those responses in terms of smoking uptake. Instead, what they have done is measured how

people who likely differ in their future propensity to smoke respond to a question about their familiarity with items featured in a particular advertising campaign.

Q. Are these problems commonly recognized by your peers?

A. Yes.

Q. But aren't these studies peer-reviewed and part of a collection of the best studies available on the issue of youth smoking and tobacco company marketing?

A. I have no reason to doubt that these studies were done with the best of intentions and may have been accepted by peers in the public health field using similar methodological approaches. These approaches, however, fall far short of those required to establish a well-founded causal relationship. These studies do not accurately model human behavior, and I would not classify these studies as careful scientific or causal analyses. Specifically, these studies ignore how human choice affects the measurement for both "treatment" and outcome. The biases that emerge from flawed modeling, measurement and interpretation are fundamental topics in my research agenda and econometrics in general. Not addressing the potential role individual choices have in shaping the choice of or acceptance of a tobacco item, or a receptivity measure, which is taken to be a surrogate for advertising, makes the studies cited in the Cochrane Review unreliable. The studies do not explicitly recognize the seriousness of these biases. Additionally, the government experts themselves do not acknowledge the obvious and severe limitations that these biases impose on any scientific inference regarding the effect of tobacco company marketing on youth smoking.

Q. Are the problems you identified recognized only in economics or are these problems recognized in other disciplines as well?

A. These problems are recognized as important limitations in other scientific disciplines as well. For example, in a 1999 article, statistician David Freedman provides numerous examples of faulty causal inference in statistical studies and discusses the "limits of current statistical techniques for making causal inferences from patterns of association." (p. 243) (Freedman, "From Association to Causation: Some Remarks on the History of Statistics," Statistical Science, Vol. 14, No. 3, p. 243-258, 1999.) (JD-013263). On page 248, Freedman also states that "...many empirical papers published today, even in the leading journals, lack a sharply focused research question; or the study design connects the hypotheses to the data collection only in a very loose way. Investigators often try to use statistical models not only to control for confounding, but also to correct basic deficiencies in the design or the data." Further, in a 2004 article, Freedman states, "As will be seen, causal relationships cannot be inferred from a data set by running regressions unless there is substantial prior knowledge about the mechanisms that generated the data."(p. 267) (Freedman, "Graphical Models For Causation, And The Identification Problem," Evaluation Review 28: 267-293, 2004.) (JD-013264).

Q. I'd like to ask you to specifically address the following studies relied upon by Dr. Eriksen in his direct testimony before the Court to support his conclusions about the relationship between tobacco company marketing and youth smoking: Pierce et. al. 1998 (JD-061624), Biener and Siegel 2000 (U.S. Ex. 72,922), Choi 2002 (US Ex. 74,019), and Sargent 2000 (JD-065832).

Have you prepared a chart outlining the primary problems with each of these studies?

A. Yes. The following chart provides a listing of these studies and the primary problems associated with using these studies to draw causal inferences regarding youth smoking and tobacco company marketing.

Study	A Principled Way for Including and Excluding Explanatory Variables in Model?	Selection Bias?	Measure of Exposure to Advertising Campaigns
Biener & Siegel 2000	No	yes	No
Pierce et. al. 1998	No	yes	No
Choi 2002	No	yes	No
Sargent 2000	No	yes	No

Q. What is your opinion about the Pierce study (JD-061624) and its reliability for drawing causal inferences about the relationship between cigarette marketing and youth smoking initiation?

A. This study purports to show evidence for a causal relationship between tobacco promotional activities and the onset of smoking. The authors studied a subset of adolescents who met the authors' criteria for being "nonsusceptible never smokers." At p.513, they found that "baseline receptivity to tobacco industry promotional activities was strongly related to which adolescents progressed toward smoking." Precisely because of the three problems I discussed above, this study does not provide any reliable evidence on the effects of tobacco advertising and non-price marketing on smoking initiation.

Q. Would you please elaborate?

A. Yes. First, the authors did not choose an estimating model on a sound statistical basis. They offer no sound theoretical or econometric basis for identifying what variables to include and exclude from the estimation analysis. For example, they do not identify whether omitted variables are potential casual factors (such as unobserved preferences) and whether these variables could be correlated with included variables.

Further, they drop variables from the analysis without explanation of whether these variables are potentially important with respect to the bias properties of the statistical approach.

This study states that: "Preliminary analyses showed no significant interactions between the index of receptivity and the exposure to smoking variables and these interactions were not retained in the final model." (p.514) This approach can lead to serious biases, because even though the eliminated variables may not be statistically significant, these variables can be correlated with both the included variables and the probability of smoking initiation. This would impose an "omitted variable bias" on the estimated specifications. Because the authors do not provide robustness checks on the results presented, it is not possible to distinguish whether the results in the Pierce study are artifacts of the particular specification they report or representative of a robust statistical association. Further, as a result of this statistical procedure, the standard errors that the authors offer are biased.

Second, the authors did not demonstrate that their receptivity measure, based on willingness to use a tobacco promotional item, possession of an item, naming an ad or having a favorite ad, is related to or correlated with *actual* advertising exposure from general tobacco advertising campaigns. Therefore, I see no reliable basis from which to conclude that they have even constructed any test of advertising exposure, let alone a flawed test. There is a crude logic to their analysis. It is that if there were no advertising whatsoever, then the various items used in some of these studies would not exist. But one must carefully distinguish two issues. The first is, are we interested in whether ownership of a specific item or "receptivity" causes smoking or whether advertising causes smoking? Second, even if we were interested in the former, this has not been established because of preference heterogeneity and self-selection.

Third, in this study receptivity simply appears to measure attitudes or preferences towards smoking. In this case, any variation in group outcomes regarding smoking initiation

would be driven by these differences in preferences. That is, adolescents likely self-select into these different receptivity groups based on their preferences and attitudes towards smoking. Because of these problems, the authors do not eliminate the possibility that their findings support only the conclusion that adolescents who have greater interest in smoking are more likely to smoke, and little else.

Q. What are your views of the reliability of the Biener and Siegel 2000 study (U.S. Ex. 72,922)?

A. The Biener and Siegel study attempts to improve upon the Pierce study I just discussed by examining the connection between advertising (using the receptivity proxy) and actual adolescent smoking behavior, instead of the relationship between advertising (using the receptivity proxy) and smoking susceptibility. They claim to find an effect of advertising, proxied by this receptivity measure, and progression to established smoking.

This study, however, also does not establish a causal inference of tobacco company advertising causing youth smoking initiation, due precisely to the problems raised in the discussion of the Pierce study: (1) the authors do not choose their estimating model on a sound statistical basis; (2) the authors did not demonstrate that their receptivity measure is related to or correlated with *actual* advertising exposure; and (3) the receptivity measure appears to be related to attitudes or preferences that influences smoking initiation.

Q. Can you be more specific?

A. Yes. Biener and Siegel, like Pierce, do not provide support for using their receptivity measure as a proxy for advertising exposure. They do not report any tests of this relationship, but simply assume this. The authors also likely create a selection bias by sorting survey

participants on the measure that they are trying to test. As I described earlier, the authors' analysis creates groups based on those who show receptivity, or an interest in smoking, which regardless of the amount of advertising exposure is more likely to lead to smoking.

Comparisons based on this type of sorting provide no necessary basis for their conclusions. As in the Pierce study discussed above, the authors provide no sound basis for the empirical model they estimate. Further, they do not provide robustness checks on these results to show how sensitive their estimates are to the seemingly arbitrary model structure imposed. An example of a robustness check would be running specifications with interactions among variables and examining the consistency and strength of the relationship of interest. One could include, for example, an interaction between parental smoking and the receptivity measure, to see if the effect of receptivity on progression to established smoking is stronger for kids with smoking parents than the average effect across all in the same receptivity category. They impose a linear model, for example, but if that is not the appropriate functional form of the underlying relationships, then the results could be biased and the effect associated with the receptivity variable could capture part of the missed explanation or inaccurate relationship imposed by the functional form. In addition, the authors choose to leave out of the specification variables that in a bivariate test are not significantly related to receptivity or progression to established smoking. This, as discussed above for the Pierce paper, could impose an omitted variable bias. Simply omitting variables that are not significant can make the final results less reliable, as well as biasing the reported standard errors.

Q. Ignoring these problems and assuming that the findings of Biener and Siegel 2000 study actually evidences a causal link between tobacco company marketing and youth

smoking initiation, would their paper allow you to assess the magnitude of such a relationship?

A. No, because they do not delineate a relationship between their receptivity measure and actual tobacco company marketing. For example, if the tobacco companies reduced their marketing expenditures in half, it is not clear what effect this would have on their receptivity measure. One could assume that this might reduce the portion of adolescents classified in the high receptivity group by a half, but this would be an arbitrary assumption and they show no relationship between their measures and volume of advertising to which the person is exposed.

Q. Another study you list is one by Sargent et al (2000) (JD-065832). Could you explain your views on this study?

A. Yes, this study looks directly at attitudes and smoking uptake--with receptivity defined as ownership or willingness to own a cigarette promotional item. At page 320, the study indicates that some students who were not receptive initially subsequently became receptive, and others who were initially classified as receptive became unreceptive. This is also reflected in the authors' conclusion, "Over time, the likelihood of smoking uptake is increased when an adolescent acquires a CPI [cigarette promotional item] or becomes willing to use one and is decreased when an adolescent who owns a CPI loses it or becomes unwilling to use it." Given the short period of time of the study (21 months), it seems questionable that changes in exposure to tobacco advertising activity itself caused the change in smoking uptake. And how can one go from being exposed to being unexposed? The changes in the receptivity variable used in their studies are likely driven not by exposure to advertising and promotions, but by attitudes or interest. Once again, the authors measure the relationship between attitudes or interest in smoking and uptake of smoking, not the effect of tobacco company advertising or promotional

activities on youth initiation, which, as I understand it, is a central issue in this case. This study does not provide evidence of such a causal link due to its theoretical and methodological shortcomings.

Q. And your views on the Choi study (U.S. Ex. 74,019)?

A. The Choi study looked at the progression to established smoking in a sample of California youth between 1993 and 1996 and its relationship to "receptivity" to tobacco advertising. Once again, the authors utilize a definition of receptivity that likely measures attitudes (high receptivity defined as recall of a favorite ad and willingness to use promotional item) rather than exposure. The authors use their conclusion of a significant relationship between receptivity and progression to established smoking to suggest a "more comprehensive restriction or complete ban on tobacco image advertising may be warranted." (p.232). This presumes that variation in the level of exposure to cigarette advertising would affect youth smoking initiation. However, this conclusion does not logically follow from the empirical findings of their study.

A difference between this study and the aforementioned studies is that it is limited to youth who were experimenters in the baseline survey. The authors acknowledge that "since this study examined only the transition from experimentation to established smoking, it is not clear whether the findings are unique to only this transition in the uptake process." (p. 232) Further, the authors themselves do not claim a causal relationship between cigarette advertising and youth smoking initiation, but simply that "receptivity to tobacco advertising and promotions predicted progression from experimentation to established smoking." (p. 231).

Q. What, if anything, do these statistical associations demonstrate with respect to causation?

A. These simple statistical associations do not support reliable evidence of any causal link between advertising and smoking initiation even for this youth subgroup. This is because of the same self-selection and measurement issues that plague the other studies. Teens who recall advertising or are willing to use cigarette promotional items likely have greater preferences for smoking, even within this "transitional" group. That is, these adolescents self-select into these different "receptivity" groups. This study also never provides any basis from which to conclude that these receptivity classifications are at all related to variation in actual exposure to advertising. Therefore, besides measuring differences in smoking preferences, these studies seem to measure little else, and thus provide no reliable evidence on the effect of advertising on youth smoking initiation.

Also, the authors provide no principled basis for selecting the empirical models they estimate, nor do they report robustness checks of their data or models. The few checks they do present indicate that even in the flawed structure of the analysis their results are not robust. Specifically, their findings show that adolescents who could name a favorite cigarette advertisement ("moderately receptive") were not statistically significantly different in smoking uptake rates from those who did not name a favorite cigarette or have a promotional item ("minimally receptive"). Therefore, the fragility of the observed relationship between their receptivity measure and youth smoking even cast into doubt whether they have statistically established a relationship between smoking preferences and smoking behavior, let alone one between advertising and smoking.

Q. Please discuss your views of Pollay, "The Last Straw? Cigarette Advertising and Realized Market Shares Among Youth and Adults." Journal of Marketing, 60(2):1-16, 1996. (U.S. Ex. 73,037).

A. Pollay did not study the impact of advertising on youth smoking initiation, although the authors could have done so with their data. Instead, they studied the relationship between brand choices and brand advertising. Brand loyalty is different than initiation. This study does not provide evidence for a causal link between tobacco advertising and youth smoking initiation, which I understand is a central issue in this case. It does not address that question. Indeed, this study doesn't even conclusively establish that youth brand choice is more sensitive to advertising expenditures than adult choice. First, the reported relationship between advertising share of voice and youth brand preference appears to be driven by one brand alone, Marlboro. Looking simply at the share of voice and realized market shares for adolescents, it appears that the ordering between share of voice and adolescent shares does not correspond, except for Marlboro. Second, it would not be surprising to find that youth would smoke a brand that is the most popular brand, in any case. There are economic incentives for individuals who begin an activity to disproportionately use common products.

In particular, popular products provide informational benefits to individuals who do not have the knowledge base yet developed to distinguish quality differentials. For example, Nobel Prize-winner Gary Becker, in his 1991 article (Becker, A Note On Restaurant Pricing And Other Examples Of Social Influences On Price, Journal of Political Economy, 99:1109-1116, 1991) suggests that restaurants may encourage long queues rather than raising prices because the enjoyment their customers get from a meal is increased by the knowledge that many other people are seeking to eat there—one can have greater confidence in the quality of the food when one observes that others also wish to purchase it. Extending this logic, one would expect a wine connoisseur to disproportionately consume esoteric labels and wine neophytes to drink more

common labels. Therefore, even if all advertisement and marketing were eliminated, one may still observe this type of correlation in the data.

Q. What are your views of the ad ban study by Saffer and Chaloupka, The Effect of Tobacco Advertising Bans on Tobacco Consumption, Journal of Health Economics, 2000(19):1117-1137 (JD-002729)? Does it provide evidence that advertising increases youth smoking?

A. The study, contrary to its stated conclusion, indicates that advertising has little or no effect on smoking behavior. The study attempts to capture variation in advertising exposure through variation in advertising restrictions. From these variations, the study draws conclusions about the effect of advertising on smoking behavior. In other words, this study captures variation in advertising exposure in a "negative" way—i.e., by reducing exposure—and thus would not be subject to the ethical concerns that the government experts say would prevent a randomized study of the positive effects of advertising.

Most ad ban studies with which I am familiar show little or no effect of advertising on smoking behavior. Even in one of the few studies to claim to find any effect, the Saffer and Chaloupka ad ban study discussed above, this effect is found not to be robust. The study by Saffer and Chaloupka examined the effect of advertising bans on cigarette consumption in 22 countries between 1970 and 1992. In this study, the authors purport to find that comprehensive bans reduce smoking. Contrary to the authors' stated conclusion, their analysis applied to all of the data they claim to draw on does not show an effect of advertising bans on cigarette or tobacco consumption. In specifications estimated on the full dataset, the coefficients on comprehensive bans are statistically insignificant across seven of the eight specifications. Indeed, in the sole specification in which the effect of bans is statistically significant for this time

period, Saffer and Chaloupka report a positive effect (244.418 with a t-statistic of 2.08), implying a comprehensive ban *increases* tobacco consumption. (See Table 4, JD-002729).

The only evidence the authors present in support of the hypothesis that bans reduce smoking come from specifications in which most of the data are excluded. Specifically, the authors summarize the results of various specifications on subsets of the data, starting in each year from 1983-1987 and ending in 1992. It is only in specifications based on these limited data that they find a negative effect of comprehensive advertising bans on cigarette consumption. Arbitrarily removing data limits observations, and therefore makes the inferences on the impact of comprehensive bans less reliable. Selecting samples to produce desired results is called data mining in statistics.

Q. Does anything else affect the reliability of Saffer and Chaloupka's results?

A. Several other factors, beyond the elimination of relevant data, affect the reliability of Saffer and Chaloupka's results. First, only two countries (New Zealand and Canada) implemented comprehensive bans between 1983 and 1992. (Saffer and Chaloupka, Tobacco Advertsing: Economic Theory and International Evidence, NBER Working Paper No. 6958, Feb. 1999). This fact implies that smoking behavior in two small countries largely drives the authors' inferences on the worldwide effects of advertising bans. Second, the authors do not include country-specific time trends to control for declines in cigarette consumption. If countries that switched to comprehensive bans during this period had already experienced steeper declines in cigarette consumption prior to implementing comprehensive bans, then this would yield a negative coefficient on the comprehensive ban variable, *even if bans had no effect on smoking behavior*. The authors also do not distinguish between the effects of bans on different types of media. For example, a ban on television advertising is weighted equally to point of purchase

advertising. Furthermore, the authors do not address how they control for taxes, restrictions on use or access to cigarettes, or other policies that may affect cigarette consumption in these countries during periods in which the bans are implemented. If these other policies are implemented simultaneously with bans, then the authors may falsely attribute to advertising bans the effects of other policies. Finally, the endogeneity of cigarette consumption and advertising bans are not accounted for in the econometric specifications. The problem is that countries with strong anti-smoking sentiment and rapidly declining cigarette consumption are the ones most likely to implement bans. Therefore, anti-smoking sentiment may be responsible for observed reductions in smoking in a country that implements a comprehensive ban and not the ban itself.

Q. Could you discuss your views on the Keeler study discussed in Keeler, et al., "The US National Tobacco Settlement: the effects of advertising and price changes on cigarette consumption," Applied Economics, 36:1623-1629 (2004)? (JD-013157)

A. The Keeler study is a time series study looking at the effect of advertising and price on per capita cigarette consumption. Therefore, it uses an econometric structure that is similar to many other econometric studies that preceded it. Many of these studies are discussed in a review by Duffy, "An Econometric Study of Advertising and Cigarette Demand in the United Kingdom," Int'l Journal of Advertising, Vol. 15, pp. 262-284 (1996). The Keeler study does not provide reliable evidence on the effect of advertising and marketing on youth smoking initiation for the following reasons: First, the study suffers from specification problems. The authors do not employ standard techniques for addressing the endogeneity problem that influences the measurement of advertising effects. Namely, changes in advertising expenditures likely are driven, at least in part, by changes in market sales. This relationship and the difficulty it imposes on measuring advertising effects were identified as a major problem in the economics literature

about thirty years ago in a 1972 book by Richard Schmalensee entitled The Economics of Advertising. This literature notes that as sales within a market increase, so do advertising expenditures, and therefore simply looking at the statistical association between advertising expenditures and cigarette sales does not allow the researcher to distinguish the directionality of the relationship. In these instances, careful economic modeling calls for the use of specific econometric tools, such as instrumental variables to distinguish whether observed relationships are at all attributable to advertising affecting consumption. The Keeler study does not employ such instruments, or any other approach, to control for the endogeneity problem associated with measuring advertising effects.

Second, the authors do not control for the timing of price changes in the actual world, but calculate an average price for the post-MSA period based on a first-stage regression and then assume that the change in the price induced by the MSA is implemented immediately after the MSA agreement. The actual price dynamic may not be accurately reflected for the short time frame where consumption and advertising are observed post MSA. This assumption can severely bias Keeler's results.

Third, the Keeler study does not explicitly examine teen smoking initiation, but rather the aggregate consumption of cigarettes for all age groups, so it does not specifically examine causal factors of smoking initiation.

VI. EVIDENCE SUPPORTS A MULTI-CAUSAL VIEW OF ADOLESCENT RISK-TAKING BEHAVIOR

Q. Please remind us of your second conclusion.

A. My second conclusion is that the literature from various disciplines supports a multi-causal view for the initiation of adolescent risk-taking behavior, and this risk-taking behavior includes youth smoking.

Q. What is the basis for your conclusion?

A. As I stated in my expert report, various disciplines, from economics to neuroscience to psychology and sociology have extensively examined and continue to examine the determinants of youth risk-taking behavior, and one conclusion is clear: there is no single-factor explanation. A consensus across the various literatures supports a multi-causal view, not only of youth smoking, but also of other risky behaviors often initiated in youth, such as drinking alcohol, smoking marijuana, engaging in unprotected sex and experimenting with hard drugs. A synthesis of studies on adolescent behavior (Kipke, Risks and Opportunities: Synthesis of Studies on Adolescence, *National Research Council and Institute of Medicine,* 1999, p. 10) (JD-012484) concludes, "Indeed, there is mounting evidence that most biological changes interact with a wide range of contextual, psychological, social, and environmental factors that affect behavior." My own research into these topics strongly supports this conclusion and augments the findings of this literature.

Even the Government's experts acknowledge that risky behaviors such as teen tobacco smoking have multiple causes. For example, in his generally excellent co-authored study of at-risk adolescents (Biglan, et al., *Helping Adolescents at Risk: Prevention of Multiple Problem Behaviors*, 2004, p. 22), Professor Biglan writes, "Documentation of the co-occurrence of problem behaviors is voluminous and growing. Numerous studies show that delinquent or antisocial behavior correlates with drug use, cigarette use, and risky sexual behavior."

Explaining youth smoking initiation, clearly, is not a simple matter, and the literature continues to advance.

Q. Could you describe some of the findings from this research on youth risk-taking behavior?

A. In this developing literature, researchers have identified a variety of "risk factors" associated with youth risky behavior but not a single unambiguous *cause* of youth risk-taking behavior. Some of this research has focused on physiological differences between youths and adults, and the role these differences play in tolerance levels and experiences of pleasure and addiction; other research has investigated social and environmental influences from families, neighborhoods and communities. These "risk factors" are described as conditions or processes that signal an increased likelihood that individuals will develop a particular behavior. (*Id.* at 60). As Biglan writes, "There is no single developmental pathway to youth problem-behavior outcomes. Therefore, no intervention will have universal effects on the reduction of these behaviors." (*Id.* at 93)

Research on adolescent risk-taking behavior identifies many of these risk factors associated with drug and tobacco use. These include individual factors, such as depression, attention deficit disorder, early and persistent antisocial, aggressive or rebellious behavior, and impulsiveness; and social influences, such as low levels of parental supervision, parental/sibling attitudes toward and use of drugs, legal penalties and sanctions, prices, and peer use of drugs. Maternal smoking is another predictor of adolescent smoking.

Researchers have found that many risk-taking behaviors are correlated with one another and this has spurred research to develop models explaining this phenomenon. A study by Morral, McCaffrey and Paddock investigated whether a common-factor cause model of "drug

use propensity" could explain both marijuana and cocaine use, in comparison to a "gateway" theory that using one drug (marijuana) acts as a gateway to using the other (cocaine). (Morral, et al., Reassessing The Marijuana Gateway Effect, RAND Drug Policy Research Center, forthcoming, p.2). Other researchers also have investigated gateway theories and presented evidence that frequent use of one drug leads to increased use of other drugs. (Pacula, "Adolescent Alcohol And Marijuana Consumption: Is There Really A Gateway Effect?" National Bureau of Economic Research, Working paper 6348 January, 1998.)

Q. What does your own research suggest on this issue?

A. My own research suggests that common factors related to ability ("cognitive factors") and to self-regulation and self-perception ("non-cognitive factors") play powerful roles in explaining a variety of behavioral problems that emerge in the adolescent years. My work builds on the current literature by pointing out that these abilities or factors are determined at very early ages of the child, long before adolescence. One of the major determinants is family -- family environments, as well as genetic factors. In "family environments" I include the in utero environment created by the mother's own behavior, such as her own smoking, drinking or stress factors operating on the fetus. The important lesson that is emerging from a variety of studies is that abilities of many sorts are shaped at early ages and they greatly affect child development and child choices to participate in risky behaviors as discussed earlier in my testimony. This is why early childhood interventions are so important.

Q. Do the government's experts agree that youth smoking initiation is likely caused by multiple factors?

A. Plaintiff's experts seem to opine that there are several causal, or in their words, "substantial contributing" factors. For example, Dr. Eriksen in his direct testimony explains that,

"Human behavior is complex and there are few if any things that are determined by only one factor." He adds that "[S]ocioeconomic status, race and ethnic classification, parental smoking, peer pressure all contribute to the initiation of smoking." (Eriksen Direct at 82, 12-13). In his co-authored study, Professor Biglan suggests that factors such as aggressive behavior in early childhood, exposure to trauma, inadequate parenting, and low socio-economic status all contribute to the likelihood that teens engage in risky behavior such as cigarette smoking. "The tendency for problem behaviors to co-occur is one of the most common findings in studies of adolescent development, but the extent to which it occurs is still not appreciated sufficiently. . . ." (Biglan et al., p. 21).

Q. Are there other causal factors related to youth smoking initiation than those mentioned by the Plaintiff's experts?

A. As I mentioned, the literature continues to develop in the area specifically addressing initiation of adolescent risky behavior, including smoking initiation. Researchers continue to investigate potential causal factors. I have looked to the findings in the literature related to smoking behavior in general for causal factors.

Q. What are some of the causal factors identified in the literature on smoking behavior in general?

A. A key finding of the economic research on smoking is that increases in the health or direct money costs of smoking lead to declines in smoking propensities. (Chaloupka and Warner, NBER Working Paper 7047, March 1999). For example, a number of studies have estimated the effect of the direct money price of cigarettes on smoking demand. One of these studies, by Lewit and Coate, "Potential for Using Taxes to Reduce Smoking," 1 Journal Health Econ. 2 (1982), 121-145) (U.S. Ex. 64,544), found that smoking by young adults (ages 20 through 24) is much

more responsive to price than smoking by older adults. Similarly, Lewit, Coate and Grossman (The Effects of Government Regulation on Teenage Smoking, 24 Journal of Law and Economics, (1981) pp. 545-569) found that propensities for adolescents (ages 12 through 17) declined with increases in cigarette prices, and that adolescent cigarette consumption was more sensitive to price than aggregate demand for other age groups. Economists have not only found that the demand for cigarettes is sensitive to current money prices, but also to anticipated future prices. (Becker, et al., An Emperical Analysis of Cigarette Addiction, 84 American Economic Review 3 (1994), 396-418 (JE-064584); Chaloupka, Rational Addiction Behavior and Cigarette Smoking, 99 Journal of Political Economy 4 (1991), 722-42) (JE-064577).

A number of studies have focused on the responsiveness of consumer demand to changes in other components of the cost of smoking, such as restrictions on access, restrictions on use, and changes in information regarding the long-term health effects of smoking. For example, restrictions on youth access to tobacco, when rigorously enforced, have been found to reduce youth smoking (Chaloupka and Warner, NBER Working Paper 7047, March 1999, at 38, citing Chaloupka and Pacula, "Limiting Youth Access to Tobacco," Working Paper, 1998 (JD-002717)). Smoking participation also has been found to respond to the release of new information regarding associated health effects, indicating that consumers take into account the health costs of smoking in deciding whether or not to smoke. Similarly, the probability of quitting has been shown to increase with the length of time an individual has smoked, which implies that smokers give greater weight to potential health costs as they become more imminent. (Douglas, The Duration Of The Smoking Habit, 36 Economic Inquiry 1 (1998), 49-64).

Q. On what basis do you conclude that price-related factors are causal factors in smoking behavior whereas you conclude there is no reliable evidence that cigarette advertising is a causal factor?

A. The studies I cited, first of all, are based on well-developed theory of consumer demand. As price or cost increases, quantity demanded decreases—this holds for *all* goods. Economic theory views the full cost to include not only direct money costs, but search costs, health costs and cost of access. The studies that I cite model the effect of price-related variables in the context of structural models that are consistent with the economic theory of consumer demand. Second, the studies employ more accurate measures of the variable of interest, price, than do the longitudinal studies cited in the Cochrane review. That is, in these studies, the variables used to proxy for cigarette prices likely capture actual price variation in the data. Third, the studies use sophisticated econometric techniques, such as instrumental variables for price, to handle the endogeneity and selection bias problem that plague the longitudinal studies cited by the government's experts. And fourth, the finding that price affects smoking behavior is robust, as it has been found in numerous types of studies.

Q. Thank you for your testimony, Dr. Heckman. Would you care to summarize your conclusions again for the Court?

A. Yes. I have two principal conclusions. First, the evidence which Plaintiff's experts present does not provide a reliable basis from which to draw informed inferences on the likely effects of tobacco company advertising and marketing on youth smoking initiation. Second, the available evidence in the developing literature on initiation of adolescent risky behavior, including smoking, supports a multi-causal model for youth smoking initiation. The factors identified in this literature as likely to be causal include parental influences, risk preferences and

peer influences. However, in this literature, cigarette advertising has not been scientifically established as a causal factor.

Q. Thank you Dr. Heckman. No further questions.